UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
PREVENTION, PESTICIDES
AND TOXIC SUBSTANCES

Note to Reader
January 15, 1998

Background: As part of its effort to involve the public in the implementation of the Food Quality Protection Act of 1996 (FQPA), which is designed to ensure that the United States continues to have the safest and most abundant food supply. EPA is undertaking an effort to open public dockets on the organophosphate pesticides. These dockets will make available to all interested parties documents that were developed as part of the U.S. Environmental Protection Agency's process for making reregistration eligibility decisions and tolerance reassessments consistent with FQPA. The dockets include preliminary health assessments and, where available, ecological risk assessments conducted by EPA, rebuttals or corrections to the risk assessments submitted by chemical registrants, and the Agency's response to the registrants' submissions.

The analyses contained in this docket are preliminary in nature and represent the information available to EPA at the time they were prepared. Additional information may have been submitted to EPA which has not yet been incorporated into these analyses, and registrants or others may be developing relevant information. It's common and appropriate that new information and analyses will be used to revise and refine the evaluations contained in these dockets to make them more comprehensive and realistic. The Agency cautions against premature conclusions based on these preliminary assessments and against any use of information contained in these documents out of their full context. Throughout this process, If unacceptable risks are identified, EPA will act to reduce or eliminate the risks.

There is a 60 day comment period in which the public and all interested parties are invited to submit comments on the information in this docket. Comments should directly relate to this organophosphate and to the information and issues available in the information docket. Once the comment period closes, EPA will review all comments and revise the risk assessments, as necessary.

These preliminary risk assessments represent an early stage in the process by which EPA is evaluating the regulatory requirements applicable to existing pesticides. Through this opportunity for notice and comment, the Agency hopes to advance the openness and scientific soundness underpinning its decisions. This process is designed to assure that America continues to enjoy the safest and most abundant food supply. Through implementation of EPA's tolerance reassessment program under the Food Quality Protection Act, the food supply will become even safer. Leading health experts recommend that all people eat a wide variety of foods, including at least five servings of fruits and vegetables a day.

Note: This sheet is provided to help the reader understand how refined and developed the pesticide file is as of the date prepared, what if any changes have occurred recently, and what new information, if any, is expected to be included in the analysis before decisions are made. **It is not meant to be a summary of all current information regarding the chemical.** Rather, the sheet provides some context to better understand the substantive material in the docket (RED chapters, registrant rebuttals, Agency responses to rebuttals, etc.) for this pesticide.

Further, in some cases, differences may be noted between the RED chapters and the Agency's comprehensive reports on the hazard identification information and safety factors for all organophosphates. In these cases, information in the comprehensive reports is the most current and will, barring the submission of more data that the Agency finds useful, be used in the risk assessments.

Jack E. Housenger, Acting Director
Special Review and Reregistration Division

TOXICOLOGY ENDPOINT SELECTION DOCUMENT

Chemical: **Fortress** (Chlorethoxyphos; DPX-43898; IN 43898) [O,O-diethyl O-(1,2,2,2-tetrachloroethyl) phosphorothioate]

PC Code: 129006
Caswell: 663P

Reviewer: Karen L. Hamernik, Ph.D. Date: 11/16/94

Section Head: Karen L. Hamernik, Ph.D. Date: 11/16/94

Branch Chief: Karl Baetcke, Ph.D. Date: 11/16/94

Dermal Absorption Data

MRID: none

% Absorbed: The dermal absorption should be considered to be 100% (dermal absorption study not available).

Supported by similarity in **Oral Lethal Dose** in female rabbits (6.7 mg/kg, MRID 408837-14 and the **Acute Dermal LD50** in female rabbits (12.5 mg/kg, MRID 408837-15).

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Acute Dietary Endpoint (One Day)

Study Selected: 6-Month Ocular Study in Beagle Dogs - No Guideline No. MRID 425592-21 Acceptable

Acute Dietary Risk Assessment Required? Yes.

EXECUTIVE SUMMARY

In an ocular toxicity study, Fortress Technical (87% - 90% a.i.) was administered in the diet to 5/sex/dose Beagle Dogs at dose levels of 0, 0.061, 0.578, or 1.880 mg/kg/day (males) and 0, 0.062, 0.619, or 1.852 mg/kg/day (equivalent to concentrations of **0, 2, 20, and 60 ppm** respectively) for 6 months (26 weeks).

There were no mortalities.

At 2 ppm, plasma cholinesterase was inhibited 12 to 21% in both sexes at day 6, and weeks 6, 13, and 26. The decrease (21%) was statistically significant in females at week 6. Inhibition at the low dose is considered to be a threshold effect of biological concern. It serves as a marker that the test material has been absorbed into the bloodstream and is of concern because the test

material is a potent cholinesterase inhibitor with a steep dose response curve.

At 20 ppm, mostly statistically significant decreases in plasma cholinesterase of 38-65% (males) and 30-63% (females) were noted at day 2, 3, and 6 and weeks 6, 13, and 26. Erythrocyte cholinesterase activity was inhibited 11-17% (mostly statistically significantly) in both sexes at weeks 6 and 13. Brain cholinesterase (cerebellum) inhibition of 19%-20% in both sexes and retinal cholinesterase inhibition of 15% (males) and 31% (females) at week 26, although not statistically significant, was considered to be of toxicologic concern.

Also at 20 ppm, a statistically significant increase in the median incidence of watery stool was noted in both sexes (for males - a median of 8 days (range 6-18 days) versus 4 in controls (range 1-7 days) and for females- a median of 6 days (range 1-63 days) versus 1 in controls (range 0-8 days). The increase was considered to be treatment-related because an alternative conclusion could not be supported due to data reporting deficiencies and discrepancies.

At 60 ppm, bilateral lacrimation was often observed in 1 female. This animal also had the lowest brain (all sites) and retinal cholinesterase activity values, relative to the rest of the high dose females, at study termination. An increased incidence of abdominal distension was noted, mostly in females (etiology unknown) in addition to further statistically significant increases in the median incidence of watery stool (for males - a median of 32 days (range 18-50 days) versus 4 in controls (range 1-7 days) and for females - a median of 21 days (range 16-82 days) versus 1 in controls (range 0-8). On days 2, 3, and 6, and weeks 6, 13, and 26 statistically significant inhibition of plasma cholinesterase activity (62-79% males and 55-80% females) was observed. Erythrocyte cholinesterase (RBC) activity was statistically significantly decreased 13-20% at weeks 6 and 13 in both sexes. [However, although a treatment-related effect on RBC cholinesterase activity (both sexes) at 20 and 60 ppm is possible, this is not clear cut because although decreases at some timepoints were statistically significant, the dose-response curves were very shallow]. Also noted at termination, were statistically significant decreases in brain cholinesterase activity (pons - 42% males, 24% females; cerebellum - 41% males, 31% females; hippocampus - 51% males, 32% females; caudate nucleus - 63% males, 47% females) and retinal cholinesterase activity (56% males, 52% females). Extraocular muscle cholinesterase activity was similar to that of controls.

The following composed the ocular assessment portion of the study: physical exam (including pupillary size and tracking , ophthalmoscopic exam, slit lamp exam, tonometry, objective refractivity, fundic photos/observations, electroretinogram

recordings (ERGs), gross necropsy, light microscopy of glutaraldehyde fixed, paraffin-embedded, hematoxylin and eosin stained sections of eye (including retina), optic nerve, and extraocular muscle, and luxol fast blue stained sections of the optic nerve. Some visual system tissue was prepared for electron microscopic exam but this was not performed. No treatment-related abnormalities were found by histopathology or most of the techniques used to assess visual system structure and function in the study. However, at 60 ppm, one female and one male had suppressed ERGs. The data were judged to be inconclusive in supporting a treatment-related effect on ERGs because the female had had some abnormal pretest readings and the findings in the male occurred at the end of the study so it could not be determined if they would have been transient.

LEL for cholinesterase inhibition following acute administration of the test material (considers plasma and RBC cholinesterase only), LEL_{acute} , is 20 ppm (0.578 mg/kg/day male, 0.619 mg/kg/day female) for cholinesterase inhibition based on plasma cholinesterase inhibition of 38% (males) and 30% (statistically significant) (females) on treatment day 2. [On day 3, plasma cholinesterase inhibition of 35 to 40% was statistically significant in both sexes at this dose]. The $NOEL_{acute}$ is 2 ppm (0.061 mg/kg/day males and 0.062 mg/kg/day females). A $NOEL/LOEL_{acute}$ for erythrocyte cholinesterase inhibition (day 2) is 60 ppm/>60 ppm (HDT), respectively.

The LEL for cholinesterase inhibition (considers all types of cholinesterase activities measured) following subchronic to chronic exposure (6 to 26 weeks), $LEL_{subchr.\ to\ chr.}$ is 2 ppm (threshold response) (0.061 mg/kg/day males and 0.062 mg/kg/day females based on plasma cholinesterase inhibition of 12 to 21% in both sexes at weeks 1, 6, 13, and 26 (statistically significant inhibition of 21% in females at week 6). A $NOEL_{subchr.\ to\ chr.}$ was not determined (although it appears to be close to 2 ppm).

The LEL for systemic toxicity is 20 ppm (0.578 mg/kg/day males, 0.619 mg/kg/day females) based on statistically significantly increased incidence of watery stool in both sexes. The NOEL is 2 ppm (0.061 mg/kg/day males, 0.062 mg/kg/day females).

The LEL for ocular toxicity (e.g. abnormal histopathology or ERG recordings; effects on ocular cholinesterase activities are treated separately) is > 60 ppm based on the absence of histopathology or clear evidence of ERG abnormalities with the techniques used. The NOEL for ocular toxicity is 60 ppm, the highest dose administered (1.880 mg/kg/day males, 1.852 mg/kg/day females).

This study is Acceptable. It satisfies the requirement for a ocular toxicity study in the dog. There is currently no guideline for this type of study. The sponsor is asked to hold onto ocular

tissues in case any further evaluation is required once guidelines are put in place.

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Note: In the 6-month ocular toxicity study in the dog, plasma and RBC cholinesterase activities were measured pretest, days 2 & 3, and weeks 1, 6, 13, 26 (non-fasted). Brain (pons, cerebellum, hippocampus, caudate nucleus) and retinal and extraocular muscle cholinesterase activities were measured at termination (fasted).
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Endpoint and dose for use in risk assessment: The critical endpoint for acute dietary risk assessment is the $NOEL_{acute}$ of 2 ppm (≈ 0.06 mg/kg/day) based on decreased plasma cholinesterase inhibition at the next higher dose (the $LOEL_{acute}$). At the $LOEL_{acute}$ of 20 ppm (0.578 mg/kg/day (males) and 0.619 mg/kg/day (females)), plasma cholinesterase activity was decreased 38% in males and 30% (statistically significant) in females on day 2 of treatment. [On day 3 of treatment, at this dose level, plasma cholinesterase was statistically significantly inhibited in both sexes by 40% (males) and 35% (females)].

Comments About Study: Inhibition of RBC cholinesterase activity did not accompany the plasma cholinesterase inhibition noted on days 2 and 3 of the study at 20 ppm. Further, there was no convincing effect on RBC cholinesterase activity during the study because although at some later timepoints, statistically significant inhibition was noted at 20 and 60 ppm, dose-response curves were very shallow.

Further dose-related increases in plasma cholinesterase inhibition (mostly statistically significant) occurred from day 6 onward at 20 and 60 ppm in both sexes.

Brain, retinal, and extraocular muscle cholinesterase activities were not measured on days 2 and 3 of the study. However, brain and retinal cholinesterase activities were inhibited at week 26 (the only measurement timepoint) in both sexes at 20 and 60 ppm. At 20 ppm, cerebellum cholinesterase inhibition of 19-20%, both sexes, and retinal cholinesterase inhibition of 15% (males) and 31% (females) were not statistically significant but were considered to be of toxicological concern since both were part of dose-related trends. At 60 ppm, further decreases of 24 to 63% were noted in cholinesterase activity in all brain areas (mostly statistically significant, both sexes) and retinal cholinesterase activity (statistically significant inhibition of 52-56%, both sexes). Extraocular muscle cholinesterase activity was not

affected.

Clinical Signs: Statistically significant dose-related increases in watery stool (otherwise unexplained) were noted in both sexes at 20 and 60 ppm at various times during the study. One 60 ppm female had treatment-related bilateral lacrimation for 20 of 26 weeks of the study.

Comment About Endpoint: When the toxicological database for Fortress is examined in its entirety, it can be seen that Fortress is a potent, highly toxic organophosphorus agent with a steep dose response curve. Females generally appear to be more sensitive than males. In some animal studies, treatment-related death was observed without accompanying clinical signs or without obvious outward signs of organophosphate toxicity. Blood cholinesterase inhibition is generally the earliest and is therefore a critical indicator that the test material has been absorbed and is interacting with target tissues. In a number of studies, brain cholinesterase inhibition is seen at or around the same dose as blood cholinesterase inhibition (including 6-Month ocular toxicity study in the dog (MRID 425592-21), 1 year dog feeding (MRID 417368-33), 90-day dog feeding (MRID 408987-03 and -04), and 90-day rat feeding (MRID 425592-15)); although, in many studies initial measurements of brain cholinesterase inhibition are performed later (i.e. 90 days, 12 months) than those for blood cholinesterase enzymes. (Other signs of toxicity may be noted at the same dose or the next higher dose). Interestingly, in one rat feeding study (MRID 412906-32) where brain cholinesterase was measured at just 6 weeks, statistically significant brain cholinesterase inhibition was observed at a lower dose (5 ppm or ≈ 0.66 mg/kg/day (females) than was plasma and RBC cholinesterase inhibition (10 ppm) (also measured at 6 weeks). See HED Document 011373, dated 12/30/94 for review of Fortress database.

Supporting Studies:

1. One Year Chronic Feeding Study in Beagle Dogs- Guideline 83-1b
MRID 417368-33
Core Guideline

Study Summary

Dietary levels: **0, 0.2, 2, 20, or 60 ppm** (0, 0.007, 0.063, 0.616 & 2.24 mg/kg/day (♂) & 0, 0.006, 0.065, 0.591 & 1.86 mg/kg/day (♀);

Plasma & RBC cholinesterase determinations were made at 1, 3, 6, 9, & 12 months & brain cholinesterase was measured at 12 months (caudate n., cerebellum/medulla, cerebrum) (fasted animals);

NOEL (ChE inhibition) = 2 ppm
0.063 mg/kg/day ♂ & 0.065 mg/kg/day ♀)
LOEL (ChE inhibition) = 20 ppm
(0.616 mg/kg/day ♂ & 0.591 mg/kg/day ♀)
based on statistically significant inhibition of plasma ChE (53-63%) (♂ & ♀) at all time points (including 1 month), RBC ChE (30-71%) (♂ & ♀) at one or more time points (better dose-response at 9 and 12 months), & cerebrum (26%) (♀) at 12 months;

At 60 ppm, plasma ChE was statistically significantly decreased 55-79% (both sexes) at all time points, RBC ChE was decreased (mostly statistically significantly) 9-67% at all time points (better dose-response at 6, 9 and 12 months in males and from 3 months on in females). Decreases in ChE activity (21-61%) were noted in all three brain areas at 60 ppm in both sexes; ChE inhibition in cerebrum and caudate nucleus was statistically significant (both sexes).

NOEL (systemic) = 20 ppm
(0.616 mg/kg/day ♂ & 0.591 mg/kg/day ♀)
LOEL (systemic) = 60 ppm
(2.24 mg/kg/day ♂ & 1.86 mg/kg/day ♀)
based on increased relative liver weight, decreased body weight gains, decreased food efficiency, decreases in RBC count, HCT, & Hb (♂), & changes in serum biochemistry parameters indicative of alterations in hepatic function (♂ & ♀). Also noted was an increase in the incidence of diarrhea (♀) and a greater duration of diarrhea per episode (♂).

Comment on Study: Same dose levels as and results generally consistent with 6-Month Ocular Toxicity Study in the dog (MRID 425592-21) discussed above.

2. 90-Day Feeding Study in Beagle Dogs Guideline 82-1(b)
MRID 408987-03, 408987-04 (dose-ranging)
Core Minimum

Study Summary

Dietary levels: **0, 0.5, 5, & 50 ppm** (0, 0.017, 0.185, 1.820 mg/kg/day (♂) & 0, 0.019, 0.186, 1.840 mg/kg/day (♀));

Plasma and RBC ChE measured twice pretest & days 45 and 90

(animals fasted); Brain ChE (caudate n., cerebellum/medulla & cerebrum) was measured at termination;

NOEL (ChE inhibition) = 0.5 ppm
(0.017 mg/kg/day)
LOEL (ChE inhibition) = 5 ppm
(0.185 mg/kg/day) based on statistically significant brain (33%) (caudate n.) (females only) at termination and plasma (38-43%) cholinesterase inhibition in both sexes at days 45 & 90; greater inhibition was seen at the high dose; RBC ChE may have been inhibited in high dose males and/or females but due to data variability even in pretest group mean values it was not clear;

NOEL (systemic) = 5 ppm
(0.185 mg/kg/day)
LOEL (systemic) = 50 ppm
(1.820 mg/kg/day ♂ & 1.840 mg/kg/day ♀) based on, in females, tremors (one female), diarrhea, transient decreases in mean body weight, elevated ALT & lower serum calcium, albumin (males also), & total protein.

Comment on Study: Doses in similar range as and results generally consistent with 6-Month Ocular Toxicity Study in the dog (MRID 425592-21) and 1 year Dog study (MRID 417368-33) discussed above.
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Short Term Occupational or Residential Exposure (1-7 Days)

Study Selected: 6-Month Ocular Study in Beagle Dogs - No Guideline No.
MRID 425592-21
Acceptable

Short Term Occupational Risk Assessment Required? Yes

Comment on Study: Same study and dose level (NOEL = 2 ppm or ≈ 0.06 mg/kg/day) as for acute dietary endpoint.

On study day 7, plasma cholinesterase was inhibited 62% in males and was statistically significantly inhibited 55% in females at the LOEL of 20 ppm (≈ 0.6 mg/kg/day). These values were part of a dose-related decreasing trend (see executive summary above for more details).

Supporting Study:

90-day Special Feeding Study in Female Rats - Guideline 82-1(a)
MRID 425592-15

Acceptable for the purposes for which it was intended (i.e. to address in particular issues of lack of NOEL for cholinesterase inhibition in the rat and the need to find or confirm a NOEL for tremor). When taken together with another rat 90-day feeding study (MRID 412906-27) and a rat 6-week feeding study (MRID 412906-32), the data are Minimum and satisfy the requirement for an 82-1a 90-day feeding study in the rat.

Study Summary

Dietary levels: **0, 0.1, 1.0, 8.0, 12.8, or 16.0 ppm** (0, 0.008, 0.080, 0.635, 1.23, & 1.63 mg/kg/day);

Plasma & RBC ChE were measured on days 1, 7, 14, 21, 28, 45, & 90 (not fasted). Brain (homogenate) ChE was measured at termination (not fasted).

NOEL (ChE inhib)$_{acute}$ = 8 ppm
(0.635 mg/kg/day)
LOEL (ChE inhib)$_{acute}$ = 12.8 ppm
(1.23 mg/kg/day) based on stat. sign. plasma ChE inhibition of 53% and RBC ChE inhibition of 13% (♀) on day 1 of treatment;

NOEL (ChE inhib)$_{subchr}$ = 1 ppm
(0.080 mg/kg/day)
LOEL (ChE inhib)$_{subchr}$ = 8 ppm
(0.635 mg/kg/day) based on stat. sign. decreases in plasma (38-46%) and/or RBC (12-21%) ChE on days 7, 14, 21, 28, 45, & 90, & brain ChE (14%) on day 90;
Further decreases in these activities were noted with increasing dose;

NOEL (systemic) = 8 ppm
(0.635 mg/kg/day)
LOEL (systemic) = 12.8 ppm
(1.23 mg/kg/day) based on mortality, clinical signs of toxicity, body weight & weight gain decreases, a reduced food efficiency. Tremor (additional observations for tremor were included in the study design) was observed in this study starting at 12.8 ppm. A dose-related increase in keratitis of the cornea was noted at the two highest dose levels (occurred mostly in moribund or animals found dead). No treatment-related retinal or optic nerve lesions were noted.

Comment on Study

On study day 7, plasma and RBC cholinesterase were statistically significantly inhibited 45% and 12% respectively (female rats) at the LOEL (ChE inhib)$_{subchr}$ of 8 ppm (0.635 mg/kg/day) with a NOEL (ChE inhib)$_{subchr}$ of 1 ppm (0.080 mg/kg/day). This NOEL is similar to that in the 6-Month Ocular Toxicity study in the dog (MRID 425592-21).

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Intermediate Term Occupational or Residential Exposure (1 Week to Several Months)

Study Selected: 6-Month Ocular Study in Beagle Dogs - No Guideline No.
MRID 425592-21
Acceptable

Intermediate Occupational Risk Assessment Required? Yes

Comment on Study: Same study and dose level (NOEL = 2 ppm or ≈ 0.06 mg/kg/day) as for acute dietary endpoint. At LOEL of 20 ppm (0.578 mg/kg/day - males and 0.619 mg/kg/day- females), there was significant (statistically or toxicologically) inhibition of plasma cholinesterase at weeks 1, 6, 13, and 26 (both sexes) and of brain (cerebellar) and retinal cholinesterase at week 26. See Executive Summary above for details.

Supporting Studies: One year dog study (MRID 417368-33) and 90-day feeding study in the dog (MRID 408987-03 and -04). See details in study summaries above.

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Cancer Classification and Basis:

Fortress is classified in Group D, "not classifiable as to human carcinogenicity, because of the inadequacy of evidence", according to the RfD/Peer Review report. An 18-month feeding carcinogenicity study in the mouse (MRID 417368-34 and -35) was negative for carcinogenicity but in a two year rat feeding study (MRID 417368-37) a slight increase in kidney tumors, not significant at $p \leq 0.05$ by the statistical methods used by the study authors, was observed in high dose group (8 ppm or 0.311 mg/kg/day) male rats. After consideration, it was decided (as stated in the RfD/Peer Review Report of Fortress, dated 3/9/95) that "the study was adequate for carcinogenicity testing" and that "due to the nature of the effect in the male rat kidney, [it was] difficult to clearly interpret the data as showing either the presence or absence of a carcinogenic effect".

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RfD and Basis:

At a meeting on November 3, 1994, the RfD/Peer Review Committee recommended an RfD of 0.0006 mg/kg/day based on an overall NOEL of 2 ppm (0.061 mg/kg/day) from the combined subchronic and chronic toxicity studies in dogs (MRIDs 425592-21, 417368-33, and 408987-03 and -04) and an uncertainty factor of 100 to account for inter-species extrapolation and intra-species variability.

MRID 425592-21 6-Month Ocular Toxicity Study in Dogs
No Guideline No.

MRID 417368-33 1-Year Chronic Feeding in Dogs
83-1b

MRID 408987-03 90-Day Feeding Study in Dogs
408987-04
82-1b

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Acute Toxicity Endpoints

The table below summarizes the results of acute toxicity studies on Fortress and the toxicity categories for the different routes of administration:

SUMMARY OF FORTRESS TECHNICAL[1] GRADE ACUTE TOXICITY DATA

Gldn	Study Type	Results	Category	MRID/HED[2]	Status[3]
81-1	Oral LD_{50} (rat-Crl:CD BR; 8 wks old)	4.8 mg/kg (♂)[6] 1.8 mg/kg (♀)	I	408837-11 (007112)	A
81-1	Oral LD_{50} (rat-Fisher 344; 10 wks old)	2.0 - 4.0 mg/kg (♂)[6] 0.5 - 2.0 mg/kg (♀)	I	408837-12 (007112)	A
81-1	Oral LD_{50} (mouse-B6C3F1)	41 mg/kg (♂)[6] 26 mg/kg (♀)	I	408837-13 (007112)	A[8]

N/A	Oral Lethal Dose (rabbit-N.Zealand White)	6.7 mg/kg (♀)[7] (estimated; only 1 animal per dose group)	I	408837-14 (007112)	N/A
81-2	Dermal LD_{50} (rabbit)	18.5 mg/kg (♂) 12.5 mg/kg (♀)	I	408837-15 (007112)	A
81-3	Inhalation LC_{50} (rat)	0.58 ppm (♂ & ♀) (0.008 mg/l)[4]	I	408837-16 (007112)	A
81-4	Prim. Eye Irrit. (rabbit)	0.1 ml too toxic to test (♂ & ♀); 2/2 deaths with 0.05 ml within 4 hrs[5]	I	408837-17 (007112)	A
81-5	Prim. Skin Irrit. (rabbit)	0.5 ml (about 200 mg/kg) too toxic to test; no dermal irrit. with 0.02 ml (about 12 mg/kg) (♂)	I	408837-18 (007112)	A
81-6	Dermal Sensitization (Guinea pig)	Buehler Method not a sensitizer using 0.4 ml of a 1% solution of technical in 80% ethanol	N/A	408837-19 (007112)	A
81-7	Delayed Neuro-toxicity (Hen)	Negative (97% TGAI or 80% TGAI)[6]	N/A	408987-02 (007112)	A

1 Test material is Fortress Technical (TGAI) (a liquid) of 86% purity unless otherwise specified; Synonyms: IN 43898, SD 208304, Chlorethoxyfos, DPX 43898
2 MRID number and (HED document number)
3 Core Grade or Acceptability Status: A = Acceptable; U = Unacceptable; N/A = Not Applicable
4 Conversion of ppm to mg/l: ppm x M.W. Fortress/24450 = 0.58 x 335.8/24450 = 0.008 mg/l
5 pH of Fortress = 3.52 $\pm$ 0.03
6 vehicle = corn oil
7 vehicle = 0.5% methylcellulose
8 Study is not Core Classified in DER but is Acceptable as an oral LD_{50} study in the mouse